experiments reported here provide further evidence of the physiological function of epinephrine in the adjustment of the organism to a situation of "stress" which involved a rapid change in the concentration of a blood electrolyte. The source of the rapidly mobilized plasma potassium after the administration of glucose *per os* and the "blocking" of this phenomenon with epinephrine is under investigation.

Summary. A hyperkalemia and cardiac standstill resulting in 100% mortality was induced within 20 minutes after the administration of 2 ml of a 50% glucose solution into the stomachs of adrenalectomized rats. Pretreatment with epinephrine 60 minutes before the administration of the glucose solution protected the adrenalectomized rats. Pretreatment with lipo-adrenal extract 120 minutes before giving the glucose solution did not protect the adrenalectomized rats. The admission of the glucose solution to demedullated rats with functional adrenal cortices resulted in a 50% mortality. The difference in mortality of the adrenalectomized and demedullated rats was ascribed to the initial relatively "low" plasma potassium concentration of the latter group. Epinephrine pretreatment protected the demedullated group too. These data are discussed as indicating that epinephrine protected the experimental groups of rats by inducing a lower than "normal" plasma potassium concentration and "blocking" a change to supranormal levels after the glucose *per os*.

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Effect of Iodide, Thiouracil and Thyroxine on the Disappearance of Thyroidal I^{131}. (18723)

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A direct method for determining *in vivo* the thyroidal secretion rate in rats has been described(1). The method consists of injecting a tracer dose of I^{131} and making daily observations over the thyroid area in terms of counts per second for a period of about 2 half-lives of I^{131} (16 days). These observations, when plotted on semilog paper, can be fitted to a straight line, and from this line the proportional rate of diminution of thyroidal I^{131} can be determined and expressed as per cent per day. This proportional secretion rate, when calculated in terms of μg of thyroxine secreted per day, agrees with calculations made by other methods. Furthermore, it has been shown that hypophysectomy, which is known to depress thyroidal activity, reduced the secretion rate to one-twelfth that of normal animals(2). Further proof that this method measures hormonal secretion was obtained by determining the effect of thiouracil, thyroxine and sodium iodide on the disappearance of thyroidal I^{131}.

Material and methods. Weanling male rats (Sprague-Dawley) were used under conditions as described previously(2). After 2 weeks on an iodine deficient diet, the animals were injected subcutaneously with a tracer dose of 1 μc of radio-iodine, and observations were made daily as described. After the 48-hour count was obtained, the animals were separated into four groups: (1) controls; (2) 1% thiouracil in the drinking water; (3) 1% sodium iodide in the drinking water; (4) 10 mg d-l-thyroxine injected daily by subcutaneous route. Observations were continued for 16 days, and the proportional rates of disappearance of thyroidal I^{131} were determined. Three experiments, performed at different times of the years, are shown in Table I.

Results. As shown in Table I, the normal rate of hormonal secretion was between 8 and 9% per day with close agreement in the 3

1. Albert, A., *Endocrinology,* 1951, v48, 334.

2. Randall, R. V., Lorenz, N., and Albert, A., *Endocrinology,* 1951, v48, 339.

TABLE I. Proportional Rate of Disappearance of Thyroidal I^{131} (Mean ± Sem.).

Exp.		Controls		Sodium iodide		Thiouracil		Thyroxine	
		No.	%/day	No.	%/day	No.	%/day	No.	%/day
1	9/1950	9	9.33 ± .49	8	4.39 ± .58	7	48.89 ± 5.80	7	4.35 ± .42
2	11/1950	9	9.25 ± .39	9	4.40 ± .45	7	56.25 ± 4.69	7	3.84 ± .39
3	1/1951	6	8.03 ± .97	7	11.52 ± 1.86	6	44.14 ± 5.00	7	4.71 ± .54

separate experiments. For thiouracil the average rate was 50% per day and for thyroxine, 4.3% per day, again with little variation among the three separate experiments. However, in the iodide experiment, the rates obtained in the first 2 experiments were distinctly different from the control rate but there was no significant change from the control rate in the third experiment.

Discussion. The results obtained add further support to the proposition that the *in vivo* method as described measures hormonal secretion and does so quantitatively. First, it is known by other tests that administration of thyroxine suppresses endogenous thyroxine secretion, and in the present experiments the suppression was more than half the control rate ($P \leqq 0.02$). Secondly, the proportional rate of disappearance (50% per day) of thyroidal I^{131} after administration of thiouracil coincides exactly with Astwood's(3) value of 50% per day for the disappearance of total thyroidal I^{127} after the administration of thiouracil.

The effect of iodide is, however, somewhat puzzling, since iodide supposedly does not affect normal thyroidal secretion. In Experiments 1 and 2, however, significant depression occurred ($P \leqq 0.01$), whereas in the third experiment, no significant change from normal was present ($P = 0.2$). These divergent results may be due (1) to the fact that iodine deficient rats are not normal rats, (2) to a seasonal effect or (3) to unknown causes. It is also puzzling that the administration of thyroxine, although inducing a significant depression of endogenous hormonal secretion, did not suppress thyroidal activity as severely as hypophysectomy did. The dose of thyroxine used was 2 to 4 times the normal daily output of thyroxine and should have induced a complete inhibition of thyroidal secretion, if its action is solely via thyrotropin inhibition. Finally, thiouracil increased the disappearance rate of thyroidal I^{131} sixfold, indicating that an immediate acceleration of TSH secretion occurred, or that a potentiating process involving TSH was invoked, or that some factor inhibiting thyroidal secretion was removed. If the effect of thiouracil were simply one of blocking further hormonal synthesis while allowing preformed hormone to be secreted at a normal rate, no effect on the disappearance rate of thyroidal I^{131} should have been noted.

Summary. The effect of thiouracil, iodide and thyroxine on thyroidal secretion was determined by an *in vivo* method utilizing I^{131}. Thiouracil accelerated thyroidal secretion sixfold; thyroxine inhibited it, and sodium iodide induced either no change or a depression of secretory rate.

3. Astwood, E. B., *Harvey Lect.*, Series 40, 1944-1945, 195.

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